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from the Author

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ON THE

NATURE AND CAUSE

OF THE

RESPIRATORY MURMUR.

BY

HYDE SALTER, M.D., F.R.S.;

FELLOW OF THE ROYAL COLLEGE OF PHYSICIANS; LECTURER ON PHYSIOLOGY AND PATHOLOGY
AT CHARING CROSS HOSPITAL MEDICAL SCHOOL; AND ASSISTANT-PHYSICIAN
TO THE HOSPITAL.

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NATURE AND CAUSE OF THE RESPIRATORY MURMUR.

The necessity of physiological as a foundation for pathological knowledge is in no way better illustrated than in the physical diagnosis of chest-disease. Not only is it necessary that we should be thoroughly acquainted with the chest-sounds in health, in order that we may have a fixed point to start from, and at once detect any aberration from their natural standard; but since morbid heart- and lung-sounds are merely modifications of the normal, and are generated by the same machinery, a knowledge of the mechanism of the sounds in health, and of the way in which the different conditions that produce them contribute to their production, is absolutely necessary, in order that we may be able not only to *detect* but to *interpret* their modifications in disease, and to perceive at once what particular aberration from the conditions of health the morbid sound we hear necessarily implies. To one who has made himself master of the *rationale* of the sounds of health, who knows exactly what produces them and how, the sounds of disease become self-interpreting—they explain themselves. Without this knowledge, they merely assert their existence, and give us no clue to their meaning; they say that something is wrong, but they do not say what, or how. I have often been struck, when listening to the morbid modifications of the respiratory murmur in disease, with the thought, how incomprehensible they would be, and how utterly they would fail to tell their story, if the *rationale* of the healthy sound was not understood. It is from considerations of this kind that many physiological questions in relation to respiration possess an interest and an importance that of themselves they would hardly seem to deserve.

One of such questions is :—What is the cause of the Respiratory Murmur ; where and how is it generated ?

That this question is a difficult one is sufficiently shown by the many answers that have been given to it ; that it is an important one every one must feel who is in the daily practice of auscultation, for it is the fundamental basis, the starting-point, from whence diverge all morbid breath-sounds, and on the views that we hold of its nature and seat must depend our interpretation of those conditions in which it is modified, obscured, or altogether lost.

It may not be amiss for me, before discussing the question, to mention a few of the opinions that have been entertained on it by distinguished authorities.

M. Beau* has advocated the view that the respiratory sound heard at the surface of the chest is not produced in the lungs, but at the pharynx, and simply depends on the transmission to the lungs of the pharyngeal sounds attending the entrance and exit of air through the pharynx.

Dr. Spittal† has advocated similar views, only he makes the seat of the sound the glottis instead of the pharynx.

Laennec considered its immediate cause to be the entrance of air into and expulsion from the air-cells.

Dr. Herbert Davies assigns the murmur to air-friction in the entire bronchial system ; he thinks the inequalities produced by the cartilaginous rings have something to do with its production ; and that the relative loudness of inspiration is due in part to the splitting of the currents of inspired air by the angles of junction of the tubes, in part to the resistance the contractile lung-tissue offers to its ingress.

Skoda is evidently of opinion that the bronchial vibrations contribute, by their conduction through the ramifying columns of air, to the respiratory murmur ; but he is not very definite, and gives no details of the mechanism of the transformation of the one sound into the other.

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* Archives Générales de Médecine, 1834.

† Edinburgh Medical and Surgical Journal, vol. xli.

Dr. Walsh attributes the sound to the entrance and exit of air to and from the air-cells, and believes that the terminal portions of the bronchial tubes are also concerned in its production. He believes also that there is a greater resistance of the textures during inspiration, and that this contributes to the excess of inspiratory over expiratory sound.

Dr. Blackiston, failing to recognise any other adequate cause for the greater loudness of inspiration than expiration, adopts the opinion that the ultimate bronchiæ contract at each inspiration, as an adjuvant power to help the air into the air-cells, and in this way produce a greater rush of air, and therefore louder sound, at inspiration ; and, relaxing at expiration, allow the air to pass noiselessly out.

In discussing this subject what we have to explain is a sound of a certain peculiar character, audible (as I am inclined to believe), in the tranquil and unconscious breathing of a healthy adult, *only at inspiration*,* but immediately becoming audible on expiration when the breathing is at all forced, and

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* In stating my belief that in the tranquil, *unconscious* respiration of health, no sound is audible at expiration, I believe I stand alone (although others admit that it is sometimes so), and in the face of the universal recognition and description of a normal expiratory murmur it may be rather rash in me to express such a belief; but the more I examine the subject the more convinced am I that such is the case; the opinion forced itself upon me, in the first place, as a result of simple observation, and in spite, of course, of my acquired opinions.

But I admit that in any case in which the normal condition of the breathing is affected by any disturbing element, expiration becomes immediately audible, and therefore that, in nineteen cases out of twenty that come under our medical observation, expiration *is* audible, because in such a percentage of cases is there some source of disturbance present. In the first line of this note it will be observed I have put the word "unconscious" in italics, and I have done so because consciousness is of itself an element of disturbance; directly the breathing becomes conscious, and the will is directed upon it, it becomes unnatural, and is carried on at too high a pitch, and expiration becomes audible. Now when a patient's chest is examined (or a person in health, it is the same thing), his attention is immediately directed to his breathing—he breathes voluntarily, and very often (with the perhaps unconscious purpose of enabling you to hear better) breathes much deeper than natural. So greatly does this element of consciousness disturb the passive tranquility of respiration, that we cannot for a single minute direct our attention to our breathing, and supervise it by our will, without inducing a peculiar kind of breathlessness, which will not subside till the attention is directed to some other subject, and respiration is left to regulate itself.

If, then, this slight exaltation of respiration, produced by the influence of the will, renders expiration audible, much more will it become so if the subject of the examination is emotionally excited, if from recent exertion or coughing the breathing is quickened, if it is embarrassed by palpitation or heart-disease, or if there is such disease of the lung elsewhere as throws on the lung at the part listened to more than its work, and renders the breathing compensatory.

But take a healthy man in a state of rest, and, while listening to his breathing, divert his attention to other subjects, or auscult him when sleeping, and I feel confident it would be very difficult to assert that any sound whatever is audible except at inspiration. Not that it is *necessary* to divert the attention in auscultating, for without doing so expiration will constantly be found soundless, but I changed in a certain way, or entirely destroyed, by certain disturbing conditions.

Our theoretical difficulty depends on our having to offer an explanation consistent with these peculiar features and circumstances of the sound; our practical difficulty depends on our being unable to imitate artificially those conditions which generate the sound in the living body. Our only elements of evidence for the solution of the problem are derived from the character and attributes of the sound itself, from a knowledge of the structure and working of the parts concerned in its generation, and from the way in which the sound is modified in disease.

One thing is certain—that the sound must be generated in the immediate neighbourhood of the superficies of the lung; its seat must be a very thin layer immediately beneath the pulmonary pleura. And why? Because it reaches the ear without conduction by the lung, and therefore must be a surface sound. That the lung is not concerned in its conduction we know, because the moment even a thin portion of the pulmonary structure acquires conducting power by condensation we hear sounds that were before inaudible. Or, stating it conversely, which perhaps may be clearer, the moment even a thin lamella of lung becomes a conductor by solidification, we hear sounds previously inaudible; the conducting power of the lung is, therefore, *nil;* the conducting power of the lung being *nil,* any healthy lung-sound must be a surface-sound; the respiratory murmur is therefore a surface-sound.*

Now, what structures have we immediately subjacent to the surface of the lung? The ultimate bronchial tubes, the intercellular passages, and the air-cells. One or all of these, then, must be the seat of the sound. Which? To answer this

am sure that when it *is* audible, it will very often become inaudible when the attention *is* so diverted.

Of course, when we are speaking of the sounds of respiration, we are speaking of them, not as they are under exceptional circumstances, but as they are as a rule, not merely when we are listening to them, but at all other times.

* I do not mean to say that the conducting power of the lung is *absolutely nil,* that the sound must be *at* the surface and no way beneath it. The effect of the non-conducting power of the lung would probably be, and no doubt is, first to make a sound muffled, and then inaudible; we do not know the exact limits beneath the surface at which a sound would cease to be heard. But we *do* know that lung condensation, sufficient to give a bronchial character to the breathing, need extend only a very little way in.

question we must understand the exact structure and relation of these parts.

The air-cells are minute alveoli, or recesses, clothing the sides and extremities of the lobular or intercellular passages. Each lobular bronchial tube, on entering its lobule, divides from four to nine times, according to the size of the lobule, and reaches the smallness of from the $\frac{1}{15}$th to the $\frac{1}{30}$th of an inch ; its branches then undergo no further reduction in size, but even enlarge a little and diverge in different directions towards the surface of the lobule, under the name of the lobular or intercellular passages ; it is these terminal air passages that are clothed with and terminate in the shallow pullulations called air-cells. The septa projecting between these air-cells, which in fact form them, are of course, like the cells themselves, shallow, and the arrangement gives to the inner surface of the lobular passage a pitted or honeycombed condition. Each ultimate element of the lung must, of course, vary in size during respiration in just the same proportion as the varying capacity of the whole organ. It has been calculated that the lung contains after an ordinary expiration, 170 cubic inches of air ; and as at each respiration 20 cubic inches of air are taken in, the capacity of the chest in ordinary breathing must vary from 170 to 190 cubic inches. Twenty of a hundred and ninety is rather more than a tenth part ; each air-cell is therefore a tenth larger at inspiration than expiration.

Now, is it conceivable that this slight variation in the capacity of these shallow open concavities should be attended with any sound ? I cannot conceive it physically possible. For be it remembered that the air-cells are not nearly-closed cavities communicating by constricted orifices with the general cavity of the lobule, but wide-mouthed and patulous, like a tea-cup. And be it remembered, too, that in respiration the air is not pumped out of and into these cells, but, as they undergo this slight change of volume, a small part of their contents passes just without them, and then again, on their recovering their capacity, from without just within them—if one can speak of 'within' and 'without' in reference to such slight interchanges of situation. For, really, the renovation of the air in the recesses of the lung does not depend on its actual removal, but upon the law of the diffusion of gases. The purpose of the movements of respiration (independent of their influence on the intrathoracic circulation) is to carry the air into bronchial tubes of a certain degree of smallness (I do not know exactly what), and, beyond that, all renovation is entrusted to diffusion action; and were it not for this the air would simply oscillate across a plane corresponding to the orifice of each alveolus, and no renovation would take place. We have only, I think, to realise what I have been endeavouring to describe, to be convinced that the air-cells themselves cannot be the seat of a breath-sound.

But, while the movement of the air at each alveolus would be so slight, so almost inappreciable, the collective expansion of all the alveoli common to a lobular passage, and the consequent abstraction of air from the general cavity, would be considerable, and would create a considerable rush of air into the lobular passage to supply its place; for the modicum of air, however small, appropriated by each dilating air-cell, would, of course, be multiplied by the number of cells communicating with the common axial cavity of the lobular passage. In the same way, the abstraction of air by all the lobular passages branching from a single ultimate bronchule would create a still more considerable abstraction of air from *it*, and, consequently, rush of air into it to supply the place of that taken. Conversely, in expiration the contribution of a tithe of their contents to the general cavity, on the part of all the alveoli common to a lobular passage would expel a considerable amount from it, and the air thus contributed by all the lobular passages common to one ultimate bronchule would occasion a still greater rush of air into and through it; and so on. In fact, the law of the branching of bronchial tubes—that the sum of the squares of the diameters of the branches is greater than the square of the diameter of the trunk from which they proceed—necessitates the continual retardation of the movement of the air in inspiration the nearer we approach the ultimate bronchial ramifications, the movement having its maximum at the trachea, and its minimum, almost perfect rest, at the plane of the orifices of the alveoli. To the same law is attributable the same result in the sanguiferous circulation, where we find the movement of the blood in the carotids to be nine inches a second, and in the capillaries $\frac{1}{30}$th of an inch in the same time. To the increasing rapid rush of air and not merely to the increasing size of the tube, is to be attributed the more and more blowing character that marks the bronchial breathing of larger and larger bronchi.

We see, then, that while we cannot refer any sound to the movement of the air in the air-cells themselves, we very reasonably can to that of the air to and from the lobular passages, still more to that in the ultimate bronchiæ, still more to that in the lobular bronchiæ. To how much of this terminal system of air-tubules are we to assign the respiratory murmur? This is a question that we do not at present possess the means of answering, because we do not at present know, as I mentioned just now, the limit of the conducting power of lung tissue. We have the results of no good experiments on this subject, nor do I think that it is one on which accurate experiments could be made, because I do not think it one in which it would be possible to imitate artificially the conditions of life. We must fall back upon clinical observation and upon the evidence afforded by critical post-mortem examinations following upon accurate auscultation during life. But this much is certain, that the lamina of lung yielding the sound must be, as I said before, exceedingly thin, and that the inaudibleness of any sound would increase in proportion to the depth from the surface. Possibly it may be restricted to the lobular passages subtending the subpleural air-cells; but I would not by any means deny the participation of the ultimate and even the lobular bronchiæ in its production, and I should not consider the non-tubularity of the sound as any evidence against this possibility, because I think it doubtful if a sound of a tubular or bronchial character could be elicited from tubes of such a degree of fineness, and because, even if it could, a sound from a single source becomes so much changed when it is multiplied by numbers, just as a single human voice differs from the hum of a crowd. But I would not say anything certain as to the distal limit of the respiratory murmur—distal, I mean, from the ear of the listener. What I wish particularly to enforce is that its proximal limit must be the lobular passages, and that the air-cells proper cannot generate it. All we know about its distal limit is, that if the bronchiæ are at all concerned in producing it (*i. e.*, immediately concerned in producing it as we hear it), clinical observation and the physics of the lungs both teach us that it can only be those of the finest calibre.

When I speak of air-cells, lobular passages, etc., of course I refer only to those of the superficies of the lung, as similar sounds generated in similar structures in deeper parts of the organ must necessarily be inaudible.

There is another reason, to which I have not referred, which makes me think that the respiratory murmur must have a tubular or quasi-tubular seat, and cannot be formed in the air-cells ; it is, that fine crepitation, such as that of pneumonia, *supplants* it ; it does not merely drown it, it supplants it, the two do not co-exist. Now, large mucous rhonchus, or subcrepitant rhonchus, or even the much smaller crepitation of the capillary bronchitis of children, does not *supplant* the respiratory murmur ; you may hear the murmur through them. These moist sounds, therefore, do not destroy the normal breath-sound, when they are not at its seat. I think we may therefore fairly conclude that a sound that *does* supplant it—that takes its place, does so because it is generated in an identical situation, and so destroys the conditions for the generation of the natural sound. I have never known true pneumonic crepitation co-exist in the same spot with the respiratory murmur, unless the crepitous patch was so small that the normal breath-sound could be conducted from the neighbourhood.

Now, those who believe that both these sounds have their seats in the air-cells, will no doubt at once adopt my conclusion (that the supplanting of the one by the other shows identity of situation), and will see in the power of pneumonic crepitation to efface the respiratory murmur an additional proof that this latter has its seat in the air-cells. But I cannot but believe that anyone who analyses the sound of crepitation, and has made himself familiar with all the grades of moist sounds, from bubbling mucous rhonchus to the crepitus of infantile capillary bronchitis, will at once perceive that these and pneumonic crepitation are sounds of identical nature ; all of them true moist sounds, depending on the interrupted passage of air through fluid, bubbling rhonchus being at one extreme of the series, and crepitation at the other. I have often been struck, when listening to the capillary crepitus of infantile bronchitis, with its close approximation, amounting almost to identity, to the fine crackle of pneumonia. In some specimens I should really have felt some difficulty in distinguishing the one from the other. In the resolution of pneumonia, too, when the exuded matter is passing along from the ultimate bronchial twigs to tubes of larger calibre, the original fine sound passes into the coarser kinds by insensible degrees, every gradation of size being filled up.

If, then, pneumonic crepitation is a veritable tube-sound, and its seat the microscopical tubes immediately subtending the air-cells, the supplanting and destruction of the respiratory murmur by it would show that this latter has an identical seat, and is therefore a tube-sound.

And now, as to the question of the actual generation of the sound, and why it is, *par excellence*, an inspiration sound.

I think there can be no doubt that the sound depends, like all healthy breath sounds (tracheal, bronchial, puerile), upon the friction of air and tube-wall, the vibrations being modified and intensified by the special acoustic properties peculiar to the tube form. The same conditions for the production of sound exist in the ultimate bronchial ramifications as in the windpipe and the larger bronchi, modified only by the extreme smallness of the tubes, and the comparative slowness of the movement of the air in these recesses of the lung.

But is the sound which is *generated* at the point at which it is audible — that is, in the sub-pleural stratum of the minutest air-tubes—the only source of the respiratory murmur? Is the air-friction in these minute tubes all that we hear? I cannot conceive but that the sound of which we know the whole bronchial system to be the seat (under the name of tracheal and bronchial breathing) must also contribute to its production. It is impossible that the vibrations in the larger trunks should not be conducted to the ultimate bronchial ramifications, and contribute their quota to the sound audible at the superficies of the chest.

For believing that these vibrations are thus conducted by the ramifying columns of air in the bronchial tubes to the chest wall, we have not only antecedent reasonableness on physical grounds; we have positive proof of it in the existence of vocal resonance and vocal fremitus; we have proof in these that the laryngeal vibrations are conducted through all the ramifications of the bronchial tree, and registered at the thoracic parietes. And if this were considered insufficient proof that the lighter and more delicate vibrations of air-and-wall friction were equally conducted, we might adduce the fact that the superaddition of the faucial and glottic sound in breathing may be distinctly appreciated at the thoracic parietes. I am quite sure that these sounds *are* audible at the surface of the chest, for I have often found them so mask the true respiratory murmur that I have been obliged to tell the patient to shut his mouth and breathe through his nose, or "cease making that hissing noise in his throat," before I could clearly hear the pure and unmixed respiratory sound. The moment, however, the patient's mouth was shut, or he ceased to breathe with a narrowed glottis, the adventitious sound was cleared away, and the natural murmur emerged in its purity. These sounds are exactly analogous in their mode of production to the tracheal and bronchial sounds (air-rush through a channel), and if *they* are conveyed by the terminal lung elements to the thoracic parietes, *à fortiori* would the bronchial sound, whose point of generation is so much nearer the seat of the respiratory murmur. I conceive it therefore impossible, in examining the causation of the respiratory murmur, and analysing the elements that contribute to it, to ignore the vibrations of which the entire bronchial system is the seat.

"But," it may be objected, "how unlike are these two sounds; how unlike are bronchial and vesicular breathing! The one harsh, tubular, and blowing; the other soft, faint, and breezy: how different their *timbre*, how different their pitch, how different their duration, and relation to inspiration and expiration!" All this I admit, and yet I think that the one enters into the composition of the other, that it furnishes a part of the basis or raw material of the sound, although so entirely modified as not to be recognisable as the same.

The modifying circumstances which, in my opinion, transform the sound we hear over a large bronchus into that which we hear in health, at every inspiration, over every part of the chest's surface, are:—

1. The reduction of the calibre of the tubes.
2. The repeated change of direction of the tubes.
3. The softening effect of the intervening film of lung.
4. The multitude of sources of sound.
5. The influence of convection in restricting the sound to inspiration.

If it can be shown that these agencies would necessarily so modify the sound as to convert the bronchial into the vesicular, it would suffice to remove the objections of those who may see in their extreme unlikeness a reason for believing that the one cannot have anything to do with the other. Let me then say a few words in explanation of how these agencies would act.

1. *Reduction of calibre.*—Sound can only be conducted unchanged as long as the conductor is uniform; when the conductor is changed, a proportionate change takes place in the sound conducted, especially if the conductor is one possessing special acoustic properties, and these are involved in the change. The conductor that, according to my theory, conducts the bronchial breath-sound to the superficies of the lung, and delivers it there as a co-efficient of the respiratory murmur, is the ramifying column of air continuous from the large bronchi to the sub-pleural air-cells. This column of air is undergoing continuous diminution, till from a diameter of $\frac{3}{4}$ of an inch, that of the windpipe, it reaches a diameter of $\frac{1}{100}$ of an inch, that, namely, of the ultimate bronchules. Each bronchial ramification that it passes through in this decreasing series has its own pitch of vibration, and will vibrate in no other; the pitch must therefore be constantly undergoing change, not being the same for the length of an inch; and in this way a good deal of what physicists call *interference* must take place, and a great consequent destruction of vibrations and loss of sound. Every day's observation, independent of theory, tells us how different is the pitch of bronchial breathing, according to the size of the tube listened to. How different, for instance, is the bronchial breathing heard over the

roots of the lungs, where the largest bronchi are, from that heard at the inferior border of the lung, when a fringe of condensation in that situation furnishes bronchial breathing of the smallest kind; the one is hollow, resonant, and large—the other small, flat, and open; the one with a tubular character, and the sound of *oo*—the other with but little tubular character, and the sound of *ah*, pronounced with the mouth wide open and the soft palate dropped a little on the root of the tongue. Now, carry this modification a little further, to the smallest tubes of all, and who shall say that the sound shall not be so changed as to reach the pitch, feebleness, and non-tubularity of the respiration-sound?

2. *Change of direction.*—But the tubes, and therefore the vibrating columns of air, are constantly, at each ramification, undergoing change of direction, and therefore reduction of sound; for the vibrations of sound, like those of light, cannot be bent out of their course without loss. This, too, would largely contribute to the enfeeblement of the bronchial sound ere it reached the terminal bronchules.

3. *The softening effect of the intervening lung-parenchyma.*—In healthy lung we never hear bronchial breathing (barring, of course, the upper sternal and interscapular sound), and when it *is* brought within the ken of the ear, it is so by some solid conductor—tubercle, or hepatised lung, for example. Not so with the respiratory murmur: *that* is so superficial, that the ear reaches it without the intervention of any solid conductor; and that the ear does so reach it, shows that the lung is not a perfect isolator of sound—that it has a certain amount of conducting power. For if it were an absolute non-conductor, the slightest removal below the pleural surface would suffice to render any sound inaudible. The effect of all imperfect isolators of sound is first to muffle and deaden the sound, and then to shut it off altogether, according to the thickness of the isolating medium. This is exactly what the lung does. Before, therefore, we reach, in passing into the lung, a point at which sound is altogether inaudible, we shall pass through a stratum in which it will be becoming increasingly indistinct. This sub-pleural stratum of lung-tissue, bounded externally by the pleura, and internally by the plane at which all sound

vanishes, is the immediate seat of the respiratory murmur, and the sound it yields must necessarily be softened and muffled by the damping influence of this thin stratum of lung.

4. *The multitude of sources of sound.*—This I am sure has a great deal to do with the soft, murmurous, rustling character of the sound, and with the strong contrast it presents to the single souffle of a bronchus.

5. *The influence of convection in restricting the sound to inspiration.*—When the intervention of a good conductor, a tubercle or hepatised lung, between the parietes of the chest and a bronchus, brings to the surface what is taking place in the tube, we hear a double blowing sound, louder in general at expiration than inspiration ; but when we listen over healthy lung, we hear that the respiratory murmur is either confined to inspiration, or, if audible at all at expiration, only faintly so. If, then, this murmur is in any degree furnished by the bronchial vibrations, how is the expiratory element destroyed while the inspiratory is retained ? By convection, and thus. In inspiration, the current of air sets towards the surface, and the bronchial vibrations are carried towards the ear of the listener ; moreover, the flow of the air surface-ward favours the propagation of the vibrations *by conduction* in this direction ; whereas in expiration the air is rushing *from* the ear of the auscultator, carrying with it the vibrations of which it is the seat, and offering an opposing force to the conduction of those vibrations towards the surface. In expiration, some of the air within the range of the ear would be transferred beyond it, and carry its vibrations with it ; in inspiration, that beyond the reach of the ear would pass the frontier, and its vibrations become audible. But I believe the influence of convection depends more upon its power of antagonising or favouring conduction, than upon its actual transfer of the vibrating air, just as a distant ring of bells will at one moment be loud, and at the next inaudible, according to the direction of the wind, though, from the rapidity with which sound is conducted, the actual movement of the air can have little to do with its conveyance.*

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* The influence of convection in determining the direction of sound is very well shown in heart-disease. In obstructive aortic disease the sound will be conveyed along the arteries, in the direction in which the blood is flowing, and inaudible over the ventricle, while, if the lesion is regurgitant, the sound, though generated at the same orifice, will be inaudible at the arteries, but conveyed back in the

Dr. Walshe assigns to convection a share in giving predominance to the inspiratory sound, but he also assigns this result in part to "the greater resistance of the textures during inspiration," an opinion in which I cannot coincide. I cannot but conceive that the air must experience *less* resistance in inspiration than expiration, for it is by the expansion of the lung and the removal of the pressure on the air, that an additional volume of air is drawn in, and it is by the resilient collapse of the lung, and the pressure it exercises on the contained air, that the expired volume is driven forth.

But is there no other cause for the relative loudness of inspiration? I think there is; and its consideration will bring me to the conclusion of this part of my subject.

In inspiration, the air is rushing from trunk to branches, and is being constantly thrown off in a new direction by the channel it is entering. In obedience to the universal law of motion, the tendency of the air rushing into the air-passages at inspiration is to move in a straight line; but it is continually deflected from the straight line by the change of direction of the tubes into which it is impelled. And what is it that so deflects it? Manifestly that side of the divergent branch it is entering on which it would impinge if it were continued in a straight line, and on which it really *does* impinge and by which it is deflected. Thus, when a main trunk divides into two branches, the central stream of air is impelled against the central spur or fork which forms the *point de depart* of the two tubes. This splits it and throws it off into two branches, part rushing into the one, and part into the other, just as the promontory of an island in the mid-stream of a river divides the water into two channels.* It is evident that here there is a more forcible impact and a more intense friction of air against the tube-wall than else-where. This will be more clearly understood by reference to the accompanying diagram. Let the two dotted lines, *x x*, represent the mid-stream of air rushing down a considerable bronchus, di-

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direction of the ventricle by the reflux of the vibrating blood. Indeed, I have sometimes heard a regurgitant aortic *bruit* clearer over the ventricle than over the orifice where it was generated.

* Dr. Quain has happily illustrated the effect of a thin edge in dividing a stream of air, and the sound produced thereby, by passing the edge of a piece of paper across a stream of air blown from the mouth.

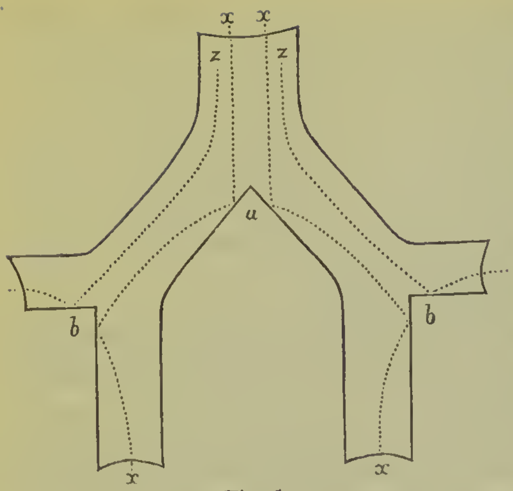

Fig. 1.

viding into two branches at *a*; it will impinge on the angle *a*, and be thrown off by its sides, and assuming a new direction, pass down the secondary bronchi accompanied by the outer portions of air which have not impinged at *a*, represented by the dotted lines *z z*: the portions of air represented by these lines, *x* and *z*, will constitute the axial column of air of the secondary bronchi, and impinge on the angles *b b*, and be by them split and thrown off into the tertiary bronchi, whose direction they will thus have acquired; and so on. This will be repeated over and over again at every branching, and thus the ingress of air into the bronchial system is a constant succession of impacts of air against the spurs or angles of tube-wall at which the tubes divaricate.

In expiration the case is very different. There the stream coming from one branch is met by the stream coming from another, and the two, reacting one upon the other, and exercising a mutual lateral pressure, pass on in an intermediate direction, the resultant of the former two; and the current tending constantly to midstream, the wall is relieved as much as possible from pressure or friction. This will be at once understoood by reference to the accompanying diagram.

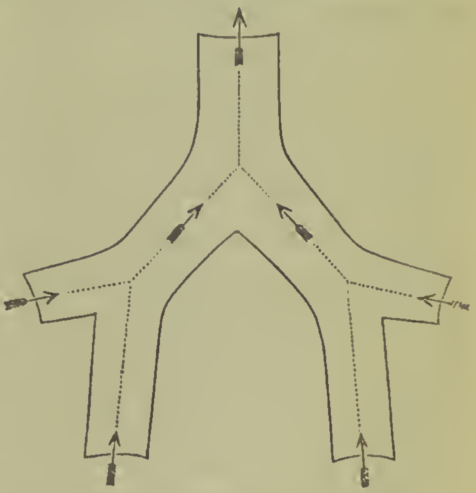

Fig. 2.

Thus, in inspiration, the stream is deflected by the resistance of the tube-wall to the moving column of air; in expiration, by the mutual lateral pressure of two columns of air moving in the same direction. There can be no doubt as to which would produce the greatest amount of friction sound.

But, it may be urged, this principle would operate equally in the largest and the smallest tubes, wherever the same principle of branching obtains; it would therefore not only be a cause tending to give predominance to inspiration in the natural surface breath-sound, but would make inspiration louder than expiration in bronchial breathing; whereas the reverse has just now been stated to be the case.

Both these facts are true; I admit them both, inconsistent as they appear. The simplest physics prove the one, and I would appeal to the experience of all observant auscultators in support of the other—whether bronchial breathing is not commonly—I do not say always—louder and more blowing at expiration than inspiration. The only way in which these two incompatible facts can be explained, is by supposing that there is some other principle in operation, some *tertium quid*, tending to augment the expiratory sound in the larger bronchial tubes, and which more than countervails the extra friction involved by the splitting of the streams of air at inspiration.

Such a principle I have long puzzled myself in search of, for I have for years noticed the relative loudness of expiration in bronchial breathing, though I have felt myself quite unable to explain it. But the other day, what I fancy must be its true explanation occurred to my mind, and though not relevant to my special object in this paper, I would suggest it to those interested in this subject for their consideration. It hinges entirely on this single anatomical fact—that the law of bronchial branching involves a continual augmentation of the aggregate volume of the tubes—that the sum of the calibre of the branches is greater than that of the trunk from which they spring. Now, if we fuse the branches, and consider them as forming a single tube, it is evident that the shape of such a tube would be that of a very elongated funnel, or cone, the narrow end of the funnel being at the windpipe, and the wide end at the periphery of the bronchial system. This is shown by the accompanying diagram (Fig. 3), where *a* represents the section of a single bronchus, and *w*, *x*, *y*, *z*, that of four in which its branching would result: it is manifest that if fused and viewed in profile, they would form a truncated cone. At

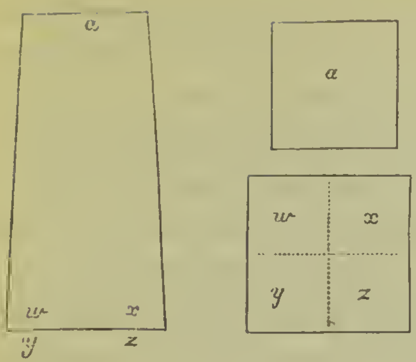

Fig. 3.

inspiration, therefore, the air would be flowing from the narrow to the wide end of the tube, and at expiration from the wide end to the narrow. The effect of this on the pressure exercised by the air on the tube walls is evident. Inspiration and expiration are like a river whose stream first widens and then contracts again. In expiration, the air is forced into what is in effect a constantly narrowing channel. As the air struggles through its straitened passage it exercises increased lateral pressure upon its walls, which react in proportion; in fact, it is this increased reaction of the walls which, together with the sustained *vis a tergo*, imparts the increased velocity to the stream of air, and makes its movement so much more rapid in the larger bronchial tubes and windpipe than in the smaller ones. In inspiration the state of things is of course exactly reversed. In the one, friction is greater than that of fluid flowing through a cylinder; in the other, it is less.

The principle *must* obtain, and of the fact endeavoured to be explained by it I have no doubt. I will leave others to determine the adequacy of the one for the explanation of the other.

There is yet one more point in which the respiratory murmur differs from bronchial breathing; it is in the relative length and rhythm of expiration and inspiration. In the respiratory murmur (when expiration *is* audible) the inspiratory sound is at least three times as long as the expiratory, and expiration succeeds inspiration so immediately that the two sounds may almost be said to be continuous; in bronchial breathing the expiratory is nearly as long as the inspiratory sound (the larger the bronchus the more equal are they), and there is an appreciable interval between them. This difference depends on these two facts—that air is an elastic fluid, and that the air in the ultimate lung structure is that nearest to, and first acted on by, the motive power—the moving thoracic parietes.

The moment the thoracic parietes, costal and diaphragmatic, cease to expand, and they and the lung are left to their tendency to collapse, expiration commences and its attendant sound, the air at once passing back from the recesses of the lung it has just entered. Not so the air in the large bronchi; that is in full inward rush when the parietes are brought to a stand and re-act on their contents. If air were perfectly inelastic, that in the large bronchi would be as instantly brought to a stand, and as instantly recoil; but being elastic, it undergoes a certain compression, the pressure re-acting on the inrushing air, first bringing it to a stop, and then driving it forth. Just at the turn of inspiration, then, the air is for an instant in a state of compression, and it is just at this moment, when the compression is turning the air in the bronchial tubes, that the pause between the inspiratory and expiratory bronchial sound occurs. The same principle that produces this pause in the smaller bronchi, of course prolongs it in the larger. Again, the sudden compression of the air which the collapse of the lung produces, is slowly dealt out in the bronchial tubes till an equilibrium of pressure is reached, and then it ceases. Thus we see that the movement of the air in the bronchial tubes is always rather behind that in the lung parenchyma, and that the "respiratory" rhythm is converted into the "bronchial" rhythm by inserting a pause between inspiration and expiration, lengthening the expiration, and shortening the post-expiratory rest.

I think I have said enough to show that the distant bronchial sound, conducted and convected to the surface by the ramifying columns of air that have a sub-pleural distribution, may contribute to the inspiration-sound of ordinary breathing; at any rate, I have stated and explained my belief, and shown that the differences of character of bronchial and vesicular breathing is no argument against the latter owing something of its raw material of sound to the former, inasmuch as those differences are just such as the modifying influences in operation would produce. If my views are accepted, an element of sound will have been added to the respiratory murmur not before indicated, and some of the difficulties of the subject will, I hope, have been cleared away.

I conclude, then—

1. That the air-cells are structurally incapable of producing a respiratory murmur by their slight dilatation.

2. That the respiratory murmur is, immediately and essentially, a fine tube sound.

3. That the lobular passages and ultimate bronchial radicles are probably its immediate seat.

4. That while the lung parenchyma, from its heterogeneous constitution, completely muffles all deep-seated sound, the unbroken column of air in the bronchial tree is an excellent conductor ; that thus the respiratory murmur has a double cause, and is in part the result of air-and-wall friction at the spot, and in part the bronchial sound of the larger tubes (although essentially modified) conducted by the unbroken column of air to the ultimate bronchial twigs.

5. That the restriction of the sound to inspiration (the "excess of inspiratory over expiratory sound" of authors) depends mainly on convection, but probably also in part on the great amount of air-and-wall friction produced on the impact of the air at the points where the tubes divaricate.

This inquiry into the nature and cause of the respiratory murmur would hardly be complete, were I not to notice a little more at length those theories of its nature and production which I have already mentioned, and demonstrate their insufficiency and untenability. I have preferred stating and supporting my own thesis first, because it will enable me so much the more concisely to point out wherein I believe these different theories are erroneous ; inasmuch as the arguments I have adduced in support of my own views will be, if considered conclusive, so many refutations of any views of a different nature ; and I shall thus merely have to refer to them instead of restating them.

Dr. Sanderson is of opinion that the only seat of the respiratory murmur is the point at which the terminal bronchial tubes end in the air-cells. His views, as far as I understand them, may be thus stated. He holds—

That the lung consists of two kinds of structures, the expansible and the non-expansible; the expansible the air-cells, the non-expansible the entire bronchial system:

That the respiratory murmur must depend upon either air and tube-wall friction, or on the movement of the air upon itself; that the latter view is not tenable, and that the former must therefore be the cause:

That the seat of the respiratory murmur will be the point where this friction is greatest; in other words, where there is the largest amount of air-movement;

That this must be the point where the non-expansible portion of the lung-structures joins the expansible; in other words, where the air-tubes end in the air-cells:

That if the air were an inelastic fluid, the greatest movement would be in the larger bronchi and windpipe, in consequence of the superficies of these tubes being less than the aggregate superficies of the bronchules in which they result; but the air being an elastic fluid, the law of "increased rapidity of stream with diminished capacity of channel" does not apply.

In an abstract of his opinions on this subject with which Dr. Sanderson has kindly furnished me, he thus explains the principles on which he arrives at his conclusions:

"At the end of the pause the air is motionless throughout the respiratory system, and its tension is zero. In inspiration the following changes take place:

"*a.* As regards tension.—An instantaneous diminution of tension is produced in the expansible portion (vesicles) of the respiratory system by the act of expansion. To restore equilibrium the tension is diminished in the inexpansible portion (bronchial tubes), but in a slighter degree, because in natural breathing, with open mouth or nostrils, the communication with the external atmosphere is so free that equilibrium is constantly maintained.

"*b.* As regards movement.—The first movement of air is from the inexpansible towards the expansible portion of the respiratory system, *i.e.* from the ultimate bronchial tubes into the air-cells. This movement, although more rapid at the first moment of the inspiratory act, is active during the whole of it. It is the commencement of a consecutive movement propagated in a direction from the smallest bronchial tubes *towards* the external orifices, *i.e.* in a direction opposite to that in which the movement itself takes place. Although *not* more rapid, it is larger than the movement that takes place in any other part of the respiratory system; that is, *more air passes from the bronchial tubes into the vesicles than enters by the mouth and nostrils.*

"The vesicular murmur is produced by this movement. Whenever air passes from a confined tube or cavity into a space of which the tension is less, a similar sound is produced.

"This movement may be called the *expansion current.* Its velocity varies—

"*Inversely,* as the quantity of air contained in the expansible portion of the lung—viz., the aggregate capacity of the vesicles;

"*Directly,* as the quantity of air inspired at each act of breathing."

While agreeing with Dr. Sanderson that the respiratory murmur depends on air- and wall-friction; that the seat of the loudest sound must be where this friction is greatest; that the air in the bronchial tubes is at one time in a state of *plus* tension, in another of *minus* tension; and that the elasticity of the air *does* modify its movements in the large and small tubes respectively, though not in the way he suggests; I object to his views, and the conclusions he draws from them, on the following grounds:

1st. That it is not correct to divide the lung into expansible and inexpansible structures—the air-cells and the air-tubes respectively; the air-tubes, especially the smaller ones, being remarkably extensible and elastic.

2ndly. That the elasticity of the air does not affect the law of the relation of rapidity of movement to calibre of tubes.

3rdly. That, in point of fact, the movement of air in the air-tubes is greatest at the windpipe, and diminishes continuously till it reaches its minimum in the ultimate bronchial tubes.

4thly. That air- and wall-friction does generate sound throughout the bronchial system, and that it is impossible but that this sound shall be conducted to the surface by those bronchial tubes that have a superficial distribution.

I admit at starting that there are some points in the physics of respiration, about which we have as yet no certain knowledge, that very materially affect this question.

One is the relative expansibility of the tubular and cellular portions of the lungs. The more the tubes expand at inspiration, the less exclusively is the twenty cubic inches of inspired air spent in the dilatation of the air-cells; and the less considerable the dilatation and change of volume of these last, the less will be the movement of the air in them and the terminal bronchules.

Another is, the average volume of the ultimate groups of air-cells and the relation of that volume, and its change in respiration, to the calibre of the terminal bronchules. This points to the same as the last proposition—the rapidity of the movement of air at the point which Dr. Sanderson believes to be the exclusive seat of the respiratory murmur.

Another is, the relative aggregate volume of the air in the air-tubes and the air in the air-cells at any one given time.

Another is, the distance down the bronchial system that the twenty cubic inches of inspired air reaches.

Another is, the law, with regard to increase of collective calibre, of bronchial branching—the ratio of increase. This would answer the last proposition: for, starting from the windpipe as a fixed point, we should soon find, if we knew the law of branching, at what point, at tubes of what calibre, the twenty cubic inches of inspired air would be used up.

Still, though these points are so uncertain, there is quite enough demonstrable and certain, in my opinion, to show that the limitation of the point of generation of the respiratory sound on which Dr. Sanderson insists, is not possible.

It seems to me that, granting the fact that the respiratory murmur depends upon the friction of air on tube or chamber wall, the questions to be answered are—"What is the seat of the greatest friction of air against the walls it rushes over?" and, "Does air-friction produce a sound in the entire bronchial system; and if so, what is to prevent its conduction to the superficies of the lung by the air in the bronchial ramifications?"

Dr. Sanderson admits that, if the air were an inelastic fluid, the whole of the traction exercised by the expansion of all the air-cells upon the air contained in all the ultimate bronchules would be transmitted undiminished to the windpipe, the single tube of supply, and produce there a much greater movement of air than that in any single ultimate bronchial tube—a movement greater by as much as the aggregate calibre of all the ultimate bronchules taken together is greater than the calibre of the windpipe. But, he says, the air is *not* an inelastic fluid, it is highly elastic, and the expansion of the air-cells is spent in diminishing the tension of the air in the peripheral portions of the bronchial system, so that the movement of the air in the ultimate bronchules is not transmitted to the larger bronchi and windpipe, or in a very diminished way, and thus that there is not that rapid current of air in the larger air-passages that there is stated to be, and that there otherwise would be. Now, if this were the case, if the movement of the air in the recesses of the bronchial system were thus lost by air-expansion from diminished tension, there would be no correspondence between the movement of air in the windpipe and that at the thoracic parietes. Whereas there is an almost exact correspondence. The expiratory and inspiratory sounds of tracheal breathing are just appreciably behind the movements of the thoracic parietes, and only just; and are slightly modified in the way and by the means I have already mentioned in a previous part of this paper. But if we listen carefully over the windpipe, we find that the inspiratory tracheal sound commences almost at the moment the thoracic parietes rise, is coincident with their movement, and ceases with it: the same at expiration; and between expiration and the next inspiration there is perfect quietness in the trachea, coinciding with the quiescence of the thoracic parietes. The same thing may be shown by the movement of air at the mouth or nostrils. It is manifest that the chink of the glottis being permanently and naturally open, and the air in the chest in communication with the external atmosphere, the air in any part of the air-tubes or at the orifices could not arrive at a state of rest until an equilibrium of tension is established: how soon such an equilibrium of tension *is* established is shown by the instantaneous rest of the air at windpipe, glottis, or nostril, that accompanies a sudden cessation of respiratory movement.

No doubt the first effect of the movement of the boundaries of the chest is to produce a state of minus tension of the intra-thoracic air at inspiration, and of plus tension at expiration; it is by this varying tension that the air in the upper parts of the bronchial system and at the orifices is moved; but the propagation is so instantaneous that the question of time may be ignored, and the air regarded as if it were inelastic. The same amount of air passes in and out of the windpipe at each inspiration and expiration as if it were absolutely inelastic. I can, in whistling, repeat a note six times in a second by six short equidistant expiratory jerks of the parietes of my chest. How could I do this if the movement of the air at the orifice of my mouth were not exactly regulable by the thoracic parietes —if the movement of the two did not coincide all but absolutely? How but for this could we ever have heard Bosio's execution of Rossini's running passages—each note isolated from its neighbour by a momentary cessation of respiratory movement?

If, then, the movement imparted to the air in the recesses of the lungs is so immediately transmitted to that of the orifices, Dr. Sanderson's theory, in principle strictly true, as far as the question of varying tension goes, in no way interferes with the law that would multiply the rapidity of the movement of the air in the windpipe as compared with that in the ultimate bronchiæ, by as much as the aggregate calibre of all these tubules exceeds that of the windpipe. The other part of his views—viz., *that more air passes from the bronchial tubes into the vesicles than enters by the mouth and nostrils,* must necessarily be erroneous, as the quantities must be exactly the same: for, until they were exactly the same, the air could not come to a condition of rest at the glottis.

We might arrive at the same conclusion another way. The quantity of air inspired at each inspiration is twenty cubic inches, or thereabouts; I have ascertained that the cubic contents of the windpipe are *six* fluid drachms, or one and a half cubic inch;* at each inspiration and expiration the air in the windpipe is, therefore, renewed fourteen times. The windpipe is three inches and a half long, therefore the air moves in the windpipe at the rate of fourteen times three inches and a half—*i.e.* forty-nine inches, each expiration and inspiration; inspiration occupying about a second and a half, and expiration less than a second. The air moves, therefore, in the windpipe at expiration at the rate of forty-nine inches—*i.e.* four feet—

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* I took every precaution to avoid fallacy in making this estimate, and was very accurate in my measurements both of the capacity and length of the windpipe. I carefully plugged each bronchus, right and left, with a cork just up to the bifurcation, and then immersed the trachea in the same fluid as that with which I filled it, in order to establish an equality of pressure inside and out, and thus preclude either distension or compression.

a second. Can it be for a moment supposed that the movement of the air at expiration in the ultimate bronchules, the movement with which it starts from a state of rest in the air-cells by the gentle collapse of the lung, is four feet a second? Such a rate of movement would carry the air from the air-cells to the orifice of the windpipe before expiration was half complete.

I think, from what I have said, that it is abundantly clear that the termination of the bronchiæ in the air-cells is not the seat of the greatest movement of air, and therefore, according to Dr. Sanderson's own postulate, not the especial seat of the generation of the respiratory sound.

I now come to the second question, "Does air-friction produce a sound in the entire bronchial system; and if so, what is to prevent its conduction of the superficies by the bronchial ramifications?" and for the answer to this question I must refer the reader to its full discussion in the preceding pages. Clinical evidence shows us that the bronchial tubes are everywhere the seat of sound, and we know that the vibration of the voice, cough, or glottic breathing, are audible at the thoracic parietes; to suppose, then, that the bronchial sound should not be audible there, would involve a suspension of physical laws.

Dr. Sanderson's theory is in part built on the supposition that the lung may be divided into an expansible and an inexpansible portion; that the air-cells are elastic and the air-tubes not. Now I am sure this is not true: I do not mean to say that the air-cells are not greatly more extensible than the tubes, though how much we do not know; but it is impossible to examine the smaller tubes minutely without seeing that they are in a high degree elastic and extensible. And remembering their constitution, we should antecedently conclude that they would be so, for their walls are largely endowed with yellow elastic fibres; indeed, the elastic coat is, with the exception of the epithelial, the only one that is continued down to the very extremities of the bronchial ramifications; so that the smallest tubes may be said to consist entirely of elastic tissue with a lining of a single layer of epithelium, and surrounded with the smallest possible quantity of connective.

Again, it is impossible to imagine the air-cells dilating without the bronchial tubes dilating too. What is it that surrounds the bronchial tubes? The parenchyma of the lung—air-cells. If we make a section of a small bronchial tube, we shall find it imbedded among air-cells, which are in contact, but not communicating, with its outer wall, just as the branches of the portal vein course their way among the lobules of the liver. Now, is it possible to imagine that these air-cells can dilate without the channel that they surround dilating also? Let A (Fig. 4) be a small bronchial tube seen in section, and B, B, B, air-cells in contact with its outer wall; the expansion of these air-cells is manifestly impossible without increasing the calibre of the cylindrical channel which they bound, unless we suppose them to expand in one direction only—that is, in a radiating direction, and not laterally, which would be gratuitous and unreasonable.

Or perhaps this may be more clear thus. Let *a* (Fig 5) be a circle formed by twelve circles, *b b b*, each of them three-quarters of an inch in diameter. The circumference of the circle will be nine inches. But if the smaller circles undergo an expansion of one-third of their diameter, so that instead of three-quarters of an inch, the diameter of each is an inch, the diameter of the whole circle which they form will have increased from nine inches to twelve. So that it is impossible for the air-cells to expand without the tubes, which are everywhere surrounded by them, expanding too, unless we suppose a vacuum to be formed; for the thin areolar web surrounding each tube, although capable of extension in any one

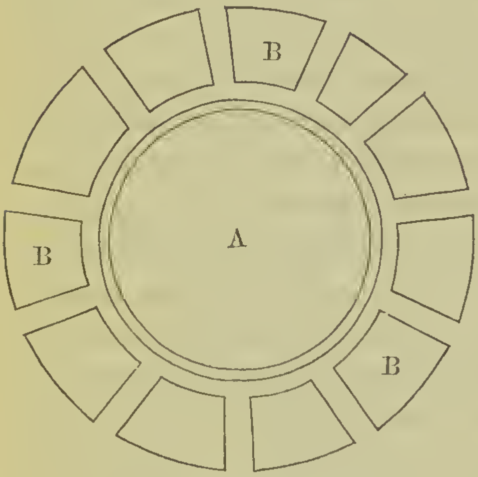

Fig. 4.

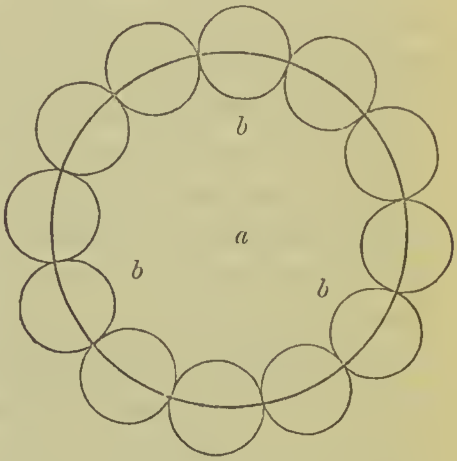

Fig. 5.

direction, is incapable of occupying any additional space. It seems to me impossible to help believing that the air-tubes *do* in this passive manner expand, the smaller ones considerably, at each inspiration.

But above all, in proof that the calibre of the bronchial tubes is increased at inspiration and diminished at expiration, we have the capital fact—a fact that I have adverted to in my work on Asthma — that one constantly finds dry musical sounds at expiration which are inaudible at inspiration ; in fact, that dry sounds are expiration sounds, and that it is the common thing to find them confined to expiration. Now, how it is possible to explain this, except by supposing that the air-tubes undergo a diminution of calibre at each expiration, I cannot imagine. On this supposition it is quite intelligible. For suppose, which is often the case, the source of the sound is a plug of tenacious mucus sticking to the side of the tube. At inspiration, when the tube is expanded in common with the other tissues of the lung, the plug of mucus does not sufficiently obstruct the air to throw it into musical vibrations; but at expiration the elastic tube, no longer expanded, is left to its own natural disposition to contract, and the plug of mucus, which in the patulous state of the tube was not equal to the production of any considerable narrowing, is now sufficient to produce a very narrow strait, perhaps almost a complete occlusion, and musical sounds are immediately generated. The mucus plug is a " constant", it cannot vary at each inspiration and expiration, and yet the conditions for the generation of the sound *do* vary, for the sound varies ; and this variation can only be produced, as I conceive, by variations in the calibre of the tube—contraction at expiration—so that what was a relatively small becomes a relatively great narrowing. If my explanation is not correct I should be glad to hear some other offered ; of the fact there can be no doubt. If my explanation *is* correct, it is a proof that the tubes *do* undergo expansion and contraction at each respiration, like the cellular structure of the lungs.

There is another fact that in my opinion tends strongly to show that the bronchial sound furnishes a good deal of the material of the respiratory murmur ; it is, the inaudibleness of the murmur at expiration. If Dr. Sanderson's view were correct, the respiratory murmur ought to be as loud at expiration as inspiration; the same amount of air rushing out of the same orifice, close to the surface, in the same time, or even less time, ought surely to make as much sound as when it rushed in. If, however, we imagine the staple of the sound to be furnished from a distance, convection comes into operation, and the audibleness of the sound will depend on the "set" of the air-current. If a bell is rung close to the ear, it makes no difference which way the wind blows, but if it is rung at a distance, we shall hear it plainly if the wind blows towards us, while when the wind blows away it may be quite inaudible. So, the fact that the direction the air is taking in the bronchial tubes determines the audibleness or inaudibleness of the respiratory murmur, shows that the sound is influencible by convection—in other words, has a distant source, and is not generated at the superficies.

I cannot therefore subscribe to Dr. Sanderson's views;

Because the air-rush, which Dr. Sanderson admits to be the cause of the sound, is at its minimum at the very part which he claims as the seat of the respiratory murmur:

Because it is impossible to conceive but that the sound generated in the entire system of air-passages must be conducted to the surface by the air in the bronchial ramifications:

Because the alternate audibleness and inaudibleness of the respiratory murmur shows it to have such a seat that its conveyance to the ear is influenced by the direction the air is taking in the bronchial tubes.

I pass now to the consideration of another set of views—those of Spittal and Beau—who transfer the seat of the respiratory murmur to the very opposite end of the air-passages, M. Beau believing that it depends on the rush of air through the pharynx, and Dr. Spittal that the chink of the glottis is its seat.

Now, with regard to the pharyngeal and glottic breath-sounds, I believe that any one may, in his own person, satisfy himself of two things:

1st. That there really are such sounds.

2nd. That they have nothing to do with the production of the ordinary respiratory murmur.

With regard to the so-called pharyngeal sound, I must premise that I do not believe it is produced in the pharynx at all, but at the isthmus faucium, by dropping the soft palate upon the root of the tongue, and so producing a narrow horizontal chink; through this the air rushes and generates the sound in question; it is of a thick, faucial, guttural character, and is the sound that marks the first heavy breathing of sleep before snoring comes on. The glottic sound is produced by the voluntary partial closure of the glottis, so narrowing the chink that the air rushes through it; it is the laryngeal element of whispering, and we hear it well in the breathing of chronic laryngitis. Each of these sounds may be produced at pleasure; we know when we are making them and when we are not; and if we listen to the chest of any one who is thus voluntarily making and unmaking them, we can tell in a moment, as I have already stated, when they are being produced and when remitted. With regard to the faucial sound, I may remark that it can never be made with the mouth shut—the glottic sound can; the opening or closure of the mouth makes no difference to it.

How, then, can these sounds have anything to do with the respiratory murmur? We cannot make and unmake it; we cannot vary it at will; we cannot affect it by anything except by the length and strength of the respiratory acts.

Dr. Walshe and Dr. Herbert Davies have attributed the excess of the inspiratory over the expiratory sound to a greater resistance offered to the air by the lung tissues in inspiration than in expiration. I must confess I am quite at a loss to comprehend the possibility of such an increased resistance. It is in consequence of the increased capacity of the air-chambers and channels of the lung that the tension of the intra-thoracic air is diminished, and the inspiratory current generated. The atmospheric pressure, balanced during a state of respiratory rest by the resistance of the walls of the air-tubes and cells, is no longer balanced; the pressure of the walls is temporarily taken off the air by their dilatation, and a state of *minus* tension is, for the moment, produced. It seems to me, then, that there must not only be not more, but less resistance to the air at inspiration than at expiration, and that this lessened resistance is the very cause of inspiration.

Dr. Herbert Davies specifies that it is the elastic contractility of the lung that offers greater resistance to the inspired air than the expired. This seems to me to imply the idea that the contractility of the lung is overcome by the distending force of the inspired air, instead of the air being drawn in in consequence of the contractile tendency of the lung being overcome by the expanding parietes of the chest. If the contractility of the lung were not, for the time, overcome, and more than overcome, no air could be drawn in. We may regard, then, the contractility of the lung at inspiration as potentially *nil.* It is to the parietes of the thoracic cavity, and to them alone, that the elasticity of the lung offers any antagonism during inspiration; and it is at expiration only, when the muscular distension of the chest ceases, that the elastic resiliency of the lung is allowed to re-assert itself, and the elastic lung-tissues re-act upon the intra-thoracic air. The resistance, therefore, offered to the air by the elasticity of the lung is an expiration and not an inspiration resistance.

In the opinion of Dr. Herbert Davies the inequalities of the internal surface of the air-passages, produced by the cartilaginous rings of the windpipe and bronchi, tend to throw the air into vibrations, and contribute to the respiratory murmur. I think it has yet to be shown that the rings of the windpipe and bronchi do produce any inequalities of surface. For my part I have never been able to see it; it has always appeared to me that the cylindrical smoothness of the internal surface is not in the slightest degree broken or interfered with by them. Indeed, to suppose that it would be, would be to suppose that Nature had been clumsy, and that she had thrown unnecessary obstruction in the way of that free flow of air into and out of the lungs, which is the essential condition of healthy breathing, and which all the other arrangements of respiration conspire to favour.

Dr. James Carson, of Liverpool, endeavoured to show, in a paper read at a meeting of the British Association for the Advancement of Science, at Manchester in 1841, that the smaller bronchial tubes contract at each inspiration, with the purpose, and with the result, of forcing the air up into their vesicular terminations; in fact, that the contraction of the bronchial tubes is an inspiratory force. Dr. Blackiston, unable to recognise in the agency of convection, or in any other way, an adequate explanation of the restriction of the respiratory sound to inspiration, adopts Dr. Carson's views, and believes that the inspiration sound is caused by the resistance which the contraction of the smaller bronchial tubes offers to the inrushing air, and that these becoming relaxed at expiration, the free and unimpeded exit of the air is permitted, and expiration is nearly or quite inaudible. Independently of these views being unsupported by any kind of evidence there are two principal objections to them, one an objection of principle, the other an objection of fact. The objection of *principle,* is that if such a contraction took place it would not be an adjuvant but an opponent force to inspiration. The respiratory murmur is co-extensive, or all but co-extensive, with the entire act of inspiration; the contraction of the bronchial tubes, therefore, which is supposed to cause it, must also continue throughout inspiration. Such a contraction, instead of assisting the air into the lungs, would simply obstruct it, and would be the most direct and efficient impediment to inspiration that could be imagined. The other objection to this theory, the objection on the score of *fact,* is comprised in the arguments against the occurrence of any rhythmical contraction of the bronchial tubes at all, either inspiratory or otherwise. There are many reasons against such a rhythmical bronchial contraction, but I think the most important and convincing one is that urged by Dr. Budd,*—that the muscles of respiration being voluntary, and the bronchial muscles involuntary, any irregularity of respiration would throw the coincidence of the two entirely out.

"It can be shown," says Dr. Budd, "that the muscular fibres of the bronchi are not muscles performing a part in the ordinary acts of breathing. Supposing them to be muscular, it is evident, from their arrangement and microscopic characters, that they belong to the muscles of organic life, or that they are involuntary muscles. But all the external muscles of respiration are voluntary muscles. Hence we shall have engaged, to accomplish the act of breathing, a voluntary and an involuntary power. The function would be easily performed as long as these powers acted in unison. But by varying the rapidity of our breathing, we should soon have two powers opposed to each other—the involuntary muscles acting to close the bronchial tubes, while the voluntary muscles acted to expand them. We should then be able to dilate the chest only when we adjusted the inspiratory movements to the action of the involuntary muscles. But we never perceive any necessity for such an adjustment. The test which this circumstance affords us is one of extreme delicacy. For, if the two powers were not exactly in unison, there would occur intervals, like the *beats* in music, when they would coincide with, or be opposed to, each other. The inspiratory acts would be alternately easy and difficult, according as the voluntary and involuntary muscles were in the same or opposite phases. But however rapid we make our breathing, we perceive no difference in the ease with which successive acts of inspiration are performed. This circumstance is a proof the most decisive, that the fibres of the bronchi have no independent rhythmical motions of contraction."

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* Medico-Chirurgical Transactions, vol. xxiii.

If, then, the bronchi do not contract at each inspiration, their contractions cannot be the cause of the respiratory murmur, and Dr. Blackiston's theory is untenable.

LONDON:
T. RICHARDS, 37 GT. QUEEN STREET. W.C.